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Electrocardiographic correlates of microalbuminuria in adult Nigerians with essential hypertension

Olusegun Busari[1], George Opadijo[2], Timothy Olarewaju[3], Abo Omotoso[4], Ahmed Jimoh[5]

[1]Department of Internal Medicine, Federal Medical Centre, Ido-Ekiti, Nigeria
[2]Faculty of Clinical Sciences, College of Medicine, Ladoke Akintola University of Teaching Hospital, Osogbo, Nigeria
[3]Clinical Research Fellow, Sheffield Kidney Institute, Northern General Hospital, Sheffield, United Kingdom
[4]Faculty of Clinical Sciences, University of Ilorin Teaching Hospital, Nigeria
[5]Department of Pathology, Federal Medical Centre, Ido-Ekiti, Nigeria

Abstract

Microalbuminuria (MA) is a predictor of excess cardiovascular morbidity and mortality in non-diabetic hypertensive patients. This study evaluated the electrocardiographic correlates of MA in adult non-diabetic Nigerians with essential hypertension. Ninety-six newly diagnosed hypertensive patients who consented and met the inclusion criteria for the study were recruited. Ninety-six age- and gender-matched normotensive controls were also studied. Resting 12-lead electrocardiogram of all patients and controls was done and the tracings analyzed by the authors for left ventricular hypertrophy with or without repolarization abnormalities, QTc prolongation, conduction abnormalities and cardiac arrhythmias such as atrial fibrillation. MA was present in 31 (32.3%) of the hypertensive patients and in only six (6.25%) of the normotensive controls. Electrocardiographic left ventricular hypertrophy (ECG LVH) was significantly more commonly found in patients with MA than in patients without it (74.2% vs *40%, p = 0.002). Left ventricular hypertrophy with ischemic pattern was significantly more frequent in the microalbuminuric hypertensive subset than in non-microalbuminuric patients (32.3%* vs *13.8%, p = 0.03). The mean QTc were 0.464 ± 0.02 s and 0.428 ± 0.017 s for microalbuminuric and non-microalbuminuric patients respectively (p = 0.01). This study shows that MA is associated with ECG abnormalities such as left ventricular hypertrophy, ischemic pattern ST-T changes and QTc prolongation. This subset of hypertensive patients constitutes a higher risk group and needs intensive monitoring and follow-up. Screening for MA should constitute part of the routine investigation of adult Nigerians with hypertension.* (Cardiol J 2010; 17, 3: 281–287)

Key words: hypertension, microalbuminuria, electrocardiographic changes, QTc

Address for correspondence: Olusegun Busari, MBBS, FMCP, Consultant Physician, Department of Internal Medicine, Federal Medical Centre, Ido-Ekiti, Nigeria, tel: +23 48 035 761 603, e-mail: olubusari@yahoo.com; olusegun_busari@yahoo.com

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Introduction

Hypertension is recognized as a common cardiovascular disease and a major risk factor for congestive heart failure, ischemic heart disease, chronic renal failure and stroke. Hypertension is defined as persistent elevation in blood pressure ≥ 140/90 mm Hg [1]. The prevalence in Nigeria, which was 15.3% (urban) and 10.6% (rural) in the general population [2, 3] was later estimated in the hospital setting to be 17–20% [4]. Cardiac damage is a common early complication of hypertension [5]. Electrocardiogram (ECG) is a useful tool to detect cardiac changes associated with hypertension [6, 7]. Microalbuminuria (MA) was first noticed in poorly controlled hypertensive individuals in 1974 [8]. MA is defined as urinary albumin excretion (UAE) of 20–200 μg/min (30–300 mg/day) or albumin creatinine ratio of 3–30 mg/mmoL (30–300 mg/g) [9]. MA is an indicator of generalized vascular damage and a marker of cardiovascular complications in hypertension [10]. This phenomenon has been associated with a higher incidence of morbid and mortal cardiovascular events [11]. The Prevention of REnal and Vascular END-stage Disease (PREVEND) study showed that MA was independently associated with ischemic ECG abnormalities [12].

MA and ECG abnormalities represent two different forms of target organ damage due to hypertension. The objective of the study was to investigate and evaluate the ECG abnormalities associated with MA in non-diabetic hypertensive adult Nigerians.

Methods

A prospective study of 96 newly diagnosed non-diabetic hypertensive patients were consecutively enrolled and compared with age- and sex-matched healthy normotensive individuals. The study was carried out between January 2003 and June 2004 at the Cardiology Unit of the University of Ilorin Teaching Hospital, Ilorin, Nigeria. Both oral and written consent was obtained from all participants and research approval was also obtained from the institutional research and ethics committee. Some exclusion criteria for the study were: previous use of antihypertensive medications, diabetes mellitus (fasting plasma glucose ≥ 7.0 mmol/L or use of insulin and/or hypoglycaemic medications) [13], renal or endocrine disease, overt proteinuria (as demonstrated by conventional dipsticks), abnormal urinary sediments on microscopy, congestive heart failure, myocardial infarction, abnormal liver function tests, obesity and use of medications that can cause ECG changes, e.g. antiarrythmic drugs, tricyclic antidepressants and macrolide antibiotics.

Clinical evaluation, definition and measurements

All participants underwent a detailed history and a thorough physical examination, including anthropometry. Laboratory assessment of cardiovascular risk factors and other relevant investigations were carried out. Body mass index (BMI) was calculated as weight (in kilograms) divided by the square of height (in meters). Obesity was defined as a BMI ≥ 30 kg/m^2 [14]. Waist-to-hip ratio was calculated as the ratio of the minimum waist circumference (a point between the upper third and the lower two thirds of the distance from umbilicus to xiphisternum) and the maximal hip circumference (the most prominent point of the gluteal region) [15].

Blood pressures were measured using a mercury column sphygmomanometer and a cuff of appropriate size. A standardized protocol was followed, in which systolic (SBP) and diastolic (DBP) blood pressures were measured on the left arm after participants had been seated for at least five minutes. Three measurements were done at least five minutes apart and the mean value used for the study. Hypertension was defined as SBP ≥ 140 mm Hg and/or DBP ≥ 90 mm Hg, or use of antihypertensive medications [1, 16, 17]. Blood samples were collected for fasting plasma glucose using the colorimetric method. Blood samples were also analyzed for serum total cholesterol (TC), triglyceride (TG), low density cholesterol (LDL) and high density cholesterol (HDL) using Rapid Analyzer 50 machine. Serum urea and creatinine and creatinine clearance were also done for all participants.

Electrocardiogram

Resting 12-lead ECG of all patients and controls were recorded using 3-channel Schiller Cardiovit-10 machine at a sensitivity of 10 mm/mV and a paper speed of 25 mm/s. Rhythm strip using standard lead II was equally done at a paper speed of 50 mm/s for QT measurement. Corrected QTc (QTc) was then derived using the Bazett's [18] formula below, where QTo is the observed QT: QTc = QTo [s]/√R-R [s].

The ECG tracings were read using a manual caliper. ECG left ventricular hypertrophy (LVH) was diagnosed using Sokolow and Lyon's criteria [19, 20]. Left atrial enlargement (LAE) was based on findings of 'P-mitrale', positive 'Morris index' [21], 'Macruz index' > 3.0 [20, 22] or any combinations of these.

Table 1. Characteristics of patients and controls.

	Patients			Controls			P
	Male (n = 52)	Female (n = 44)	Total (n = 96)	Male (n = 49)	Female (n = 47)	Total (n = 96)	
Mean age (year)	51.2 ± 10.1	48.2 ± 8.8	49.7 ± 12.7	49.6 ± 12.3	42.6 ± 10.4	46.1 ± 13.0	0.2
Mean SBP [mm Hg]	164.5 ± 14.2*	155.5 ± 15.1*	160.0 ± 15.0	132.4 ± 9.7*	125.6 ± 8.2*	129.0 ± 10.0	0.01
Mean DBP [mm Hg]	111.5 ± 10.1*	103.3 ± 11.8	107.4 ± 10.5	82.1 ± 5.9*	78.3 ± 7.1	80.2 ± 6.9	0.01
Mean PP [mm Hg]	53.0 ± 10.7	52.2 ± 12.9	52.6 ± 8.6	50.3 ± 6.5	47.3 ± 7.6	48.8 ± 7.1	0.1
TC [mmol/L]	4.55 ± 0.76*	4.37 ± 0.81*	4.46 ± 0.79	3.50 ± 0.41*	3.28 ± 0.38*	3.39 ± 0.42	0.02
TG [mmol/L]	1.28 ± 0.27	1.24 ± 0.29	1.26 ± 0.32	1.23 ± 0.12	1.17 ± 0.11	1.20 ± 0.18	0.4
LDL [mmol/L]	3.29 ± 0.68*	3.11 ± 0.79*	3.20 ± 0.77	2.52 ± 0.42*	2.36 ± 0.39*	2.44 ± 0.38	0.03
HDL [mmol/L]	1.10 ± 0.22*	1.02 ± 0.26*	1.06 ± 0.25	1.27 ± 0.17*	1.21 ± 0.19*	1.24 ± 0.13	0.05
CL [mL/min]	81.5 ± 19.4	73.3 ± 16.7	77.42 ± 20.72	88.7 ± 14.2	79.9 ± 13.8	84.28 ± 17.10	0.06
CR [μmol/L]	95.9 ± 12.1	90.9 ± 13.2	93.4 ± 11.5	88.7 ± 10.6	85.3 ± 11.2	87.0 ± 10.8	0.5

*p < 0.05; SBP — systolic blood pressure; DBP — diastolic blood pressure; PP — pulse pressure; TC — total cholesterol; TG — trigliceride; LDL — low density protein; HDL — high density protein; CL — creatine clearance; CR — creatinine

Microalbuminuria testing

MA was determined using the Micra Test II test strips (Boehringer Manneheim GMBh, Mannheim, Germany). This dipstick has been found to be the fastest, most accurate and, relatively, cheapest way to screen patients for the presence of MA [23]. There are four color blocks on the test strip corresponding to negative (or 0), 20, 50 and 100 mg/L of albumin. The test was done on three consecutive first morning voided urine samples collected at three weekly intervals. MA was considered to be present when two of the three urine samples tested produced a reaction color corresponding to 20 mg/L or more. The mean value of the MA was also recorded for each participant.

Data analysis

The data collected was analyzed with SPSS 10.5 software. Percentages and proportions were used to describe categorical variables while means and standard deviations were used for numerical variables. Student's t test and χ^2 were used to analyze differences between variables as appropriate. $P < 0.05$ was taken as indicating statistical significance.

Results

The prevalence of MA in the hypertensive group was 32.3% and 6.25% in the controls. The mean age (±SD) of the patients was 49.7 ± 12.7 years with a range of 24–77 years and the male to female ratio was 1.34:1.0. The mean age (±SD) of the controls was 46.1 ± 13 years with a range of 20–74 years. There was no statistically significant difference between the mean ages (±SD) of patients and controls (p = 0.06). There was also no statistically significant difference between the mean ages (±SD) of patients with MA and those without MA (52.5 ± 11.9 *vs* 48.3 ± 13.0, p = 0.1). Table 1 enumerates other characteristics of the patients and the controls. Patients with MA had statistically significant higher DBP (p = 0.03) than those without MA. However, the difference between SBP (p = = 0.07) and pulse pressure (PP) (p = 0.06) was not statistically significant (Table 2). MA patients also had significantly lower HDL and higher TC and LDL than their counterparts without MA. But the means of TG were not statistically different between MA and non-MA groups. Patients with MA were more likely to have ECG LVH than patients without it (74.2% *vs* 40%). LVH with ischemic pattern was significantly more common in MA hypertensive patients than those without MA (32.3% *vs* 13.8%, p = 0.03; Table 3). Although 13 (41.9%) MA patients had LAE, there was no significant difference when compared to non-MA patients (p = 0.06). But when compared to the controls, LAE was significantly more common (p = 0.0001; Table 4). Five (16.1%) patients with MA and the same number without MA (7.7%) had QTc prolongation (p = 0.01). The means QTc were 0.464 ± 0.02 s and 0.428 ± ± 0.017 s for MA and non-MA patients respectively (Table 4). Arrhythmias such as atrial fibrillation,

Table 2. Characteristics of patients with and without microalbuminuria (MA).

	Patients with MA			Patients without MA			P
	Male (n = 18)	Female (n = 13)	Total (n = 31)	Male (n = 37)	Female (n = 18)	Total (n = 65)	
Mean age (year)	54.9 ± 8.8	50.6 ± 9.2	52.5 ± 11.9	49.5 ± 10.1	47.8 ± 8.7	48.3 ± 13.0	0.1
Mean SBP [mm Hg]	185.2 ± 18.4	179.5 ± 20.1	182.2 ± 20.4	162.8 ± 17.9	168.1 ± 18.3	168.3 ± 22.1	0.07
Mean DBP [mm Hg]	120.3 ± 20.5*	119.7 ± 17.0*	120.5 ± 18.7	100.8 ± 15.1*	100.4 ± 12.1*	102 ± 14.9	0.03
Mean PP [mm Hg]	64.7 ± 10.9	80.1 ± 12.4	81.8 ± 11.7	60.3 ± 13.1	66.5 ± 105	66.4 ± 10.2	0.06
TC [mmol/L]	5.05 ± 0.87*	4.94 ± 0.78*	5.0 ± 0.56	4.11 ± 0.62*	3.99 ± 0.53*	4.05 ± 0.5	0.04
TG [mmol/L]	1.47 ± 0.21	1.35 ± 0.24	1.41 ± 0.35	1.39 ± 0.25	1.25 ± 0.29	1.32 ± 0.29	0.2
LDL [mmol/L]	4.08 ± 0.51*	3.90 ± 0.49*	3.99 ± 0.49	2.52 ± 0.42*	2.36 ± 0.39*	2.44 ± 0.38	0.001
HDL [mmol/L]	0.93 ± 0.18*	0.89 ± 0.17*	0.91 ± 0.16	1.27 ± 0.17*	1.21 ± 0.19*	1.24 ± 0.13	0.01
CL [mL/min]	68.7 ± 12.5*	62.3 ± 10.8*	63.6 ± 11.5	79.7 ± 13.1*	71.5 ± 11.4*	72.5 ± 12.6	0.02
CR [μmol/L]	96.4 ± 14.1	95.9 ± 13.7	94.3 ± 10.6	94.1 ± 13.2	93.1 ± 12.9	93.5 ± 11.7	0.5

*p < 0.05; SBP — systolic blood pressure; DBP — diastolic blood pressure; PP — pulse pressure; TC — total cholesterol; TG — trigliceride; LDL — low density protein; HDL — high density protein; CL — creatine clearance; CR — creatinine

Table 3. ECG chamber hypertrophy and QTc in patients with and without microalbuminuria (MA).

	Patients with MA			Patients without MA			P
	Male (n = 18)	Female (n = 13)	Total (n = 31)	Male (n = 37)	Female (n = 18)	Total (n = 65)	
LVH with ischemic pattern	7 (38.9%)*	3 (23.1%)	10 (32.3%)	5 (13.5%)*	4 (22.2%)	9 (13.8%)	0.03
LVH without ischemic pattern	7 (38.9%)*	6 (46.2%)	13 (41.9%)	8 (21.6%)*	9 (50.0%)	17 (26.2%)	0.01
LAE	11 (61.1%)*	2 (15.4%)*	13 (41.9%)	9 (24.3%)*	6 (33.3%)*	15 (23.1%)	0.06
BAE	2 (11.1%)*	—	2 (6.5%)	1 (2.7%)*	—	1 (1.5%)	
Mean QTc	0.471 ± 0.014*	0.460 ± 0.019	0.464 ± 0.02	0.435 ± 0.01*	0.0426 ± 0.13	0.428 ± 0.017	0.001
Number with QTc prolongation	4 (22.2%)*	1 (7.7%)	5 (16.1%)	3 (8.1%)*	2 (11.1%)	5 (7.7%)	0.01

*p < 0.05; LVM — left ventricular hypertrophy; LAE — left atrial enlargement; BAE — biatrial enlargement

Table 4. ECG chamber hypertrophy and QTc in patients with microalbuminuria (MA) and controls.

	Patients with MA			Controls			P
	Male (n = 18)	Female (n = 13)	Total (n = 31)	Male (n = 49)	Female (n = 47)	Total (n = 96)	
LVH with ischemic pattern	7 (38.9%)	3 (23.1%)	10 (32.3%)	—	—	—	
LVH without ischemic pattern	7 (38.9%)*	6 (46.2%)*	13 (41.9%)	5 (10.2%)*	5 (10.6%)*	10 (10.4%)	0.07
LAE	11 (61.1%)*	2 (15.4%)*	13 (41.9%)	2 (4.1%)*	1 (2.1%)*	3 (3.1%)	0.0001
BAE	2 (11.1%)	—	2 (6.5%)	—	—	—	
Mean QTc	0.471 ± 0.014*	0.460 ± 0.019*	0.464 ± 0.02	0.392 ± 0.18*	0.410 ± 0.02*	0.395 ± 0.012	0.001
Number with QTc prolongation	4 (22.2%)*	1 (7.7%)	5 (16.1%)	1 (2.0%)*	1 (2.1%)	2 (2.1%)	0.006

*p < 0.05; LVM — left ventricular hypertrophy; LAE — left atrial enlargement; BAE — biatrial enlargement

Table 5. Arrhythmias and heart blocks in patients with and without microalbuminuria (MA).

	Patients with MA			Patients without MA			P
	Male (n = 18)	Female (n = 13)	Total (n = 31)	Male (n = 37)	Female (n = 18)	Total (n = 65)	
AF	1 (5.6%)	—	1 (3.2%)	—	1 (5.6%)	1 (1.5%)	
PAC	2 (11.1%)	1 (7.7%)	3 (9.7%)	3 (8.1%)	2 (11.1%)	5 (7.7%)	0.6
PVC	6 (33.3%)*	2 (15.4%)	8 (25.8%)	3 (8.1%)*	1 (5.6%)	5 (7.7%)	0.03
1st degree AVB	1 (5.6%)	1 (7.7%)	2 (6.4%)	1 (2.7%)	3 (16.7%)	4 (6.2%)	0.4
2nd degree AVB	1 (5.6%)	—	1 (3.2%)	—	—	—	
3rd degree AVB	—	—	—	—	—	—	
LAHB alone	2 (11.1%)	2 (15.4%)	4 (12.9%)	5 (13.5%)	3 (16.7%)	8 (12.3%)	0.4
LPHB alone	1 (5.6%)	—	1 (3.2%)	—	—	—	
RBBB	1 (5.6%)	1 (7.7%)	2 (6.4%)	2 (5.4%)	3 (16.7%)	5 (7.7%)	0.2
LBBB	3 (16.7%)	1 (7.7%)	4 (12.9%)	3 (8.1%)	—	3 (4.6%)	0.05
LAHB + RBBB	1 (5.6%)	—	1 (3.2%)	1 (2.7%)	—	1 (1.5%)	
LPHB + RBBB	—	—	—	—	—	—	
TFB	—	—	—	—	—	—	

*p < 0.05; AF — atrial fibrillation; PAC — premature atrial complex; PVC — premature ventricular complex; AVB — atrioventricular block; LAHB — left anterior hemiblock; LPHB — left posterior hemiblock; RBBB — right bundle branch block; LBBB — left bundle branch block; TFB — trifasciular block

premature ventricular complex (PVC) and premature atrial complex (PAC) were more frequent in patients with MA than in those without MA (Table 5). PVC was significantly more frequent in MA than non-MA patients (p = 0.04). Intraventricular conduction abnormalities were more common in patients with MA than their counterparts without MA and the controls (Table 5). Left bundle branch block was present in four (12.9%) and three (4.6%) MA and non-MA patients respectively (p = 0.05). Although PAC (9.7% *vs* 2.1%, p = 0.2), and most of the heart blocks i.e. 1st degree atrioventricular block (6.4% *vs* 4.2%, p = 0.4), left anterior hemiblock (12.9% *vs* 5.2%, p = 0.06), right bundle branch block (6.4% *vs* 4.2%, p = 0.4), were more frequent in patients with MA than in the controls, the differences were not significant.

There was a significant positive correlation between MA and DBP (r = 0.38, p = 0.018), QTc (r = 0.67, p = 0.01), LVH (r = 0.42, p = 0.021), TC (r = 0.51, p = 0.019) and LDL (r = 0.49, p = 0.020). A significant negative correlation existed between MA and HDL (r = –0.27, p = 0.037) and creatinine clearance (r = –0.24, p = 0.040); while no significant relationship was found with regard to age, SBP (r = 0.22, p = 0.055), PP (r = 0.14, p = 0.089), TG (r = 0.11, p = 0.094) and creatinine (r = 0.73, p = 0.296).

Discussion

This study has shown that MA is associated with ECG abnormalities and other risk factors for cardiovascular morbidity and mortality in non-diabetic adult Nigerians with hypertension. This accords with previous research [10–12, 24–26]. The prevalence of MA (32.3%) is not too far from the 25.4% found in a South African study [27]. However, it is important to acknowledge that the settings of the two studies were different. Patients with MA had significantly higher DBP than those without MA, and DBP was positively and significantly correlated with MA. This finding is at variance with the study by Akinsola et al. [24] which reported a significant positive correlation between MA and SBP and not DBP. Although SBP has remained a more consistent determinant of MA in hypertensives, some other studies have also implicated DBP and PP. A study of 539 Chinese women and 795 men showed that DBP was independently correlated with UAE [28, 29].

In this study, hypertensive patients with MA were more likely to have ECG LVH than both the patients without MA and the normotensive controls. In fact, ECG LVH with ischemic pattern was significantly more common in the subset with MA. There was also a significant positive correlation between MA and LVH. This finding was similar to the report of PREVEND study which showed that MA was independently associated with ischemic electrocardiographic abnormalities in a large non-diabetic population [12]. Also, Salles et al. [30] found that the presence of ECG strain was associated with higher 24-hour MA. ECG LVH has been shown to be an independent risk factor for sudden cardiac death [24]. This may partly explain the excess car-

diovascular morbidity and mortality associated with MA in non-diabetic hypertensive patients. In fact, Dell'omo et al. [31] concluded in their study that LVH explained, at least in part, the predictive power of MA for morbid events. The conferment of greater cardiovascular risk when LVH is associated with ST-T wave changes has been documented in many studies [32, 33]. Opadijo et al. [33] concluded that LVH with ST-T wave changes signified a higher coronary risk in adult Nigerians.

It is also evident from this study that patients with MA had significantly longer mean QTc than their counterparts without MA. QTc prolongation was more frequent in the former than in the latter group. QTc also showed significant positive correlation with MA. The significance of this finding is that hypertensive patients with MA are more likely to develop arrhythmias that are associated with QTc prolongation such as PVC and ventricular tachycardia. It also suggests that MA has additional value to conventional risk indicators in predicting cardiovascular events in non-diabetic hypertensive patients. Patients with MA are also more likely to have PVC than patients without MA. Major ECG abnormalities in hypertension may be as a result of pressure overload and atherosclerotic vascular damage and are considered predictors of future vascular events [34]. Pontremoli et al. [35] also revealed that MA has a significant and independent role in target organ damage, particularly ECG abnormalities.

Our study also revealed that patients with MA had significantly higher TC and LDL, and lower HDL, than patients without MA and the controls. MA was positively and significantly correlated with TC and LDL and negatively and significantly correlated with HDL. This finding is consistent with the reports of previous studies. Biggazi et al. [36] reported that dyslipidemia was associated with MA and added to the cardiovascular risk in hypertension.

Conclusions

MA signifies ECG abnormalities such as ECG LVH, QTc prolongation and ventricular arrhythmias in adult non-diabetic Nigerians with hypertension. It indicates excess cardiovascular morbidity and mortality. Hence the subset of adult Nigerian hypertensive patients with MA constitutes a higher risk group and needs close monitoring and follow-up. These patients may also benefit from antihypertensive drugs that have been proven to be effective in reducing albuminuria. We recommend routine screening for MA in newly diagnosed hypertensive patients and at regular intervals during follow-up.

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